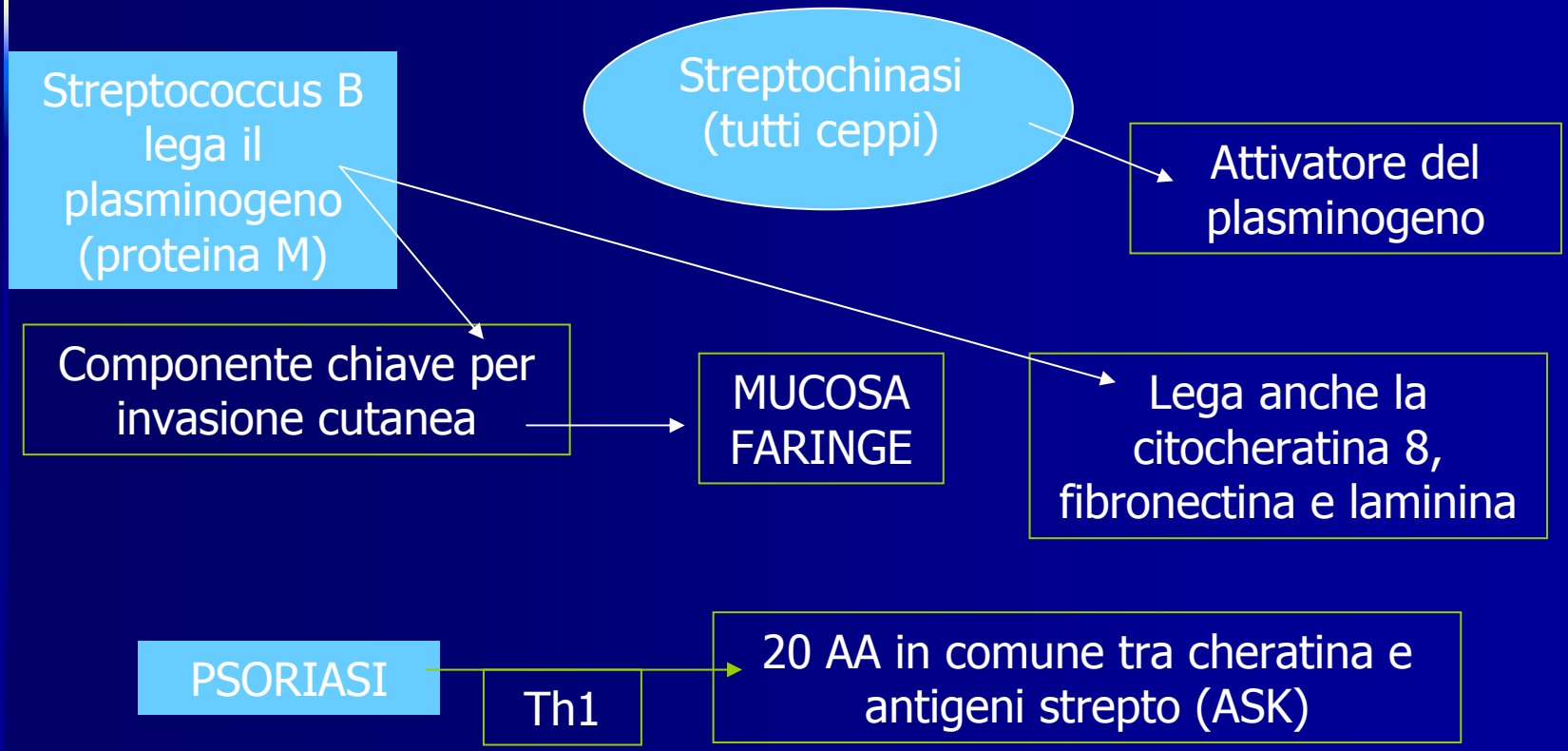
DALLA PSORA AL KRÄTZE

Elementi di base per la comprensione dell'eczema

Agente psorico



Tamura GS, Nittayajarn A. Group B streptococci and other gram-positive cocci bind to cytokeratin 8. Infect Immun. 2000 Apr;68(4):2129-34.

Group B streptococci (GBS) aderisce ai recettori di superficie, presenti sulle cellule epiteliali; questi recettori includono fibronectin and laminin, cytokeratin 8 (CK8).

CK8 solubile lega anche Staphylococcus aureus, Lactococcus lactis, Enterococcus faecalis, Streptococcus pyogenes.

Adesione di GBS alla cytokeratin può essere importante per il mantenimento della colonizzazione nei siti di epitelio cheratinizzato, quali vagina, o per l'adesione di tali batteri alle cellule epiteliali danneggiate in altri siti

Dallo SF, et al. Biofunctional domains of the Mycoplasma pneumoniae P30 adhesin. Infect Immun. 1996 Jul;64(7): 2595-601.

Esistono epitopi condivisi tra P30 adhesin and fibrinogen, keratin, and myosin. Ciò rinforza l'importanza di P30 nella citoadesione e nella virulenza, oltre a porre una base molecolare, per l'autoimmunità post-infettiva, associata e mediata da Mycoplasma pneumoniae.

Geyer A, et al. M protein of a Streptococcus dysgalactiae human wound isolate shows multiple binding to different plasma proteins and shares epitopes with keratin and human cartilage. FEMS Immunol Med Microbiol. 1999 Oct;26(1):11-24.

Accanto al gruppo A (GAS), il gruppo C di Lancefield dei beta-emolitici (GCS) sono implicati nella patogenesi delle faringiti purulente

cross-reactivity between a 68-kDa cartilage protein

also cross-reacted with antibodies recognising epidermal keratins

Our data, obtained with MC, suggest that not only infections with GAS but also infections with GCS and possibly GGS may be responsible for the formation of streptococcal-associated sequel diseases.

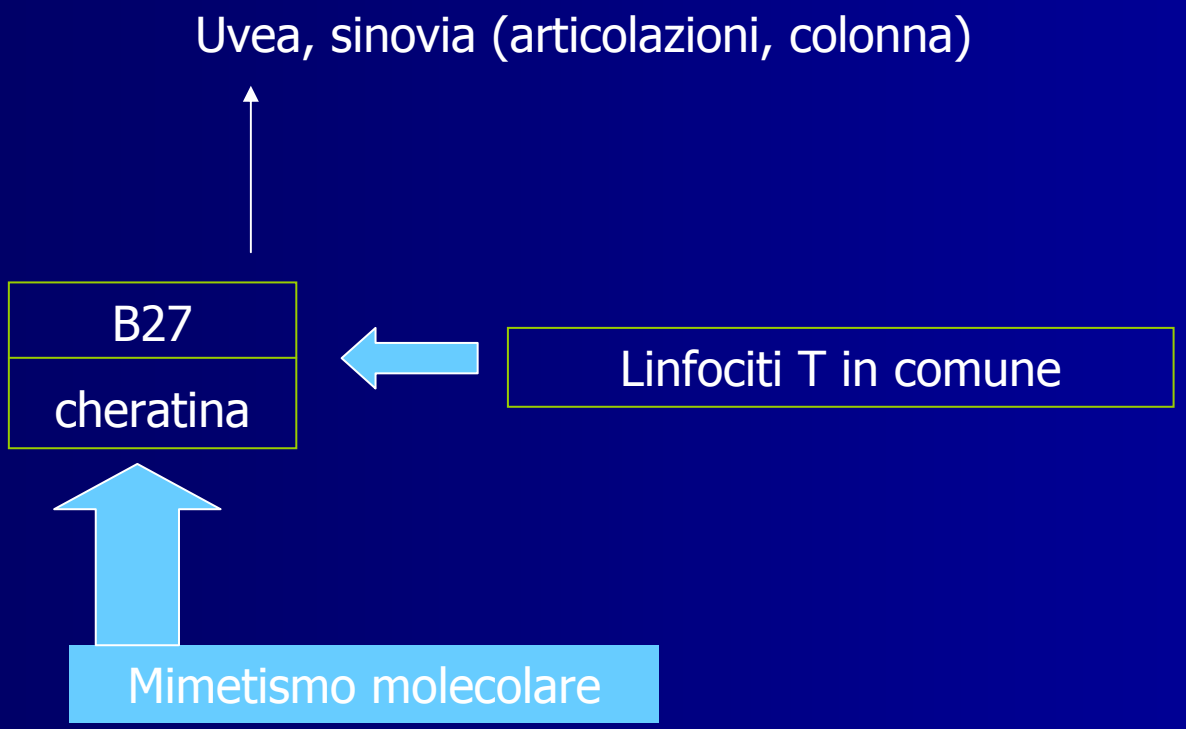
Hikhman AR, et al. A subset of mouse monoclonal antibodies cross-reactive with cytoskeletal proteins and group A streptococcal M proteins recognizes N-acetyl-beta-D-glucosamine. J Immunol. 1993 Oct 1;151(7):3902-13

anticorpi N-acetyl-beta-D-glucosamine (GlcNAc) mostrano reazione crociata con valvole cardiache, cute e altri tessuti.

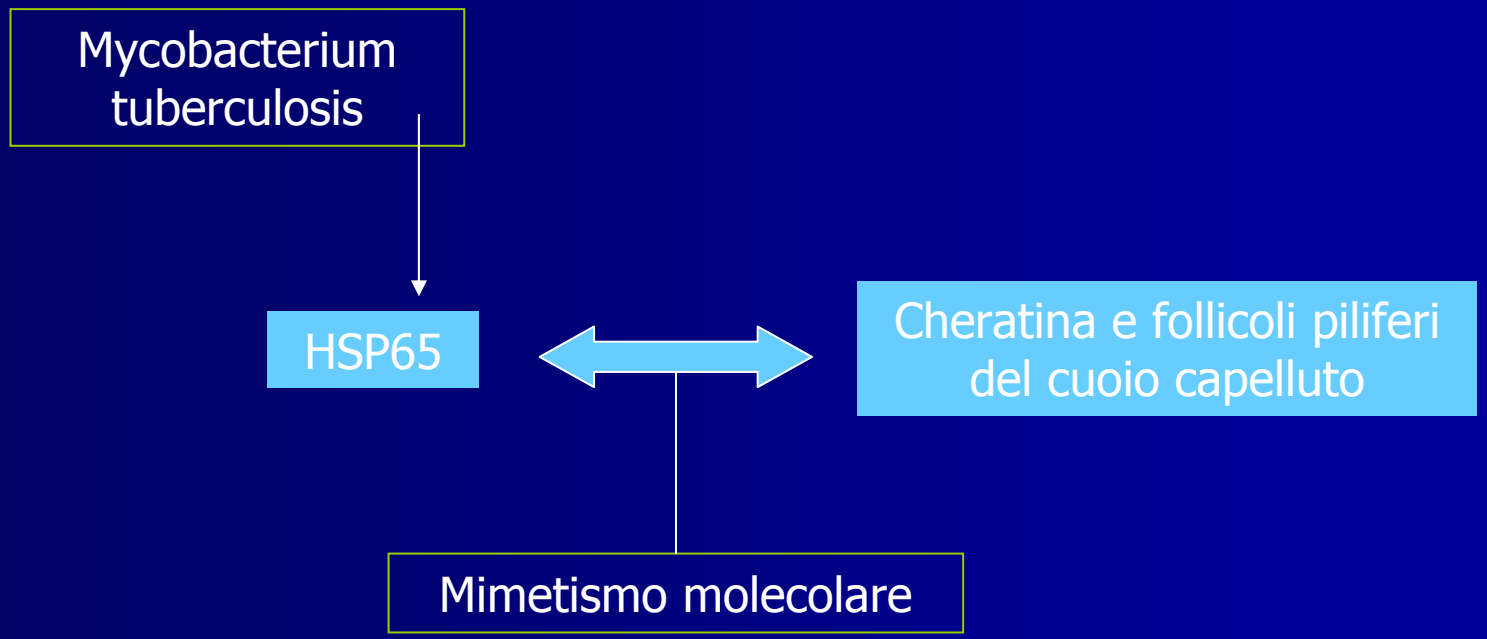
one of the mAb, 49.8.9, was found to react most strongly with a synthetic peptide sequence synthesized from the coxsackievirus B3 capsid protein VP1, which shows homology with and cross-reacts with sequences in the streptococcal M6 protein and human cardiac myosin

This most interesting mAb, previously shown to neutralize coxsackie viruses, recognized the amino acid sequence RRKLEFF, which may mimic the GlcNAc epitope.

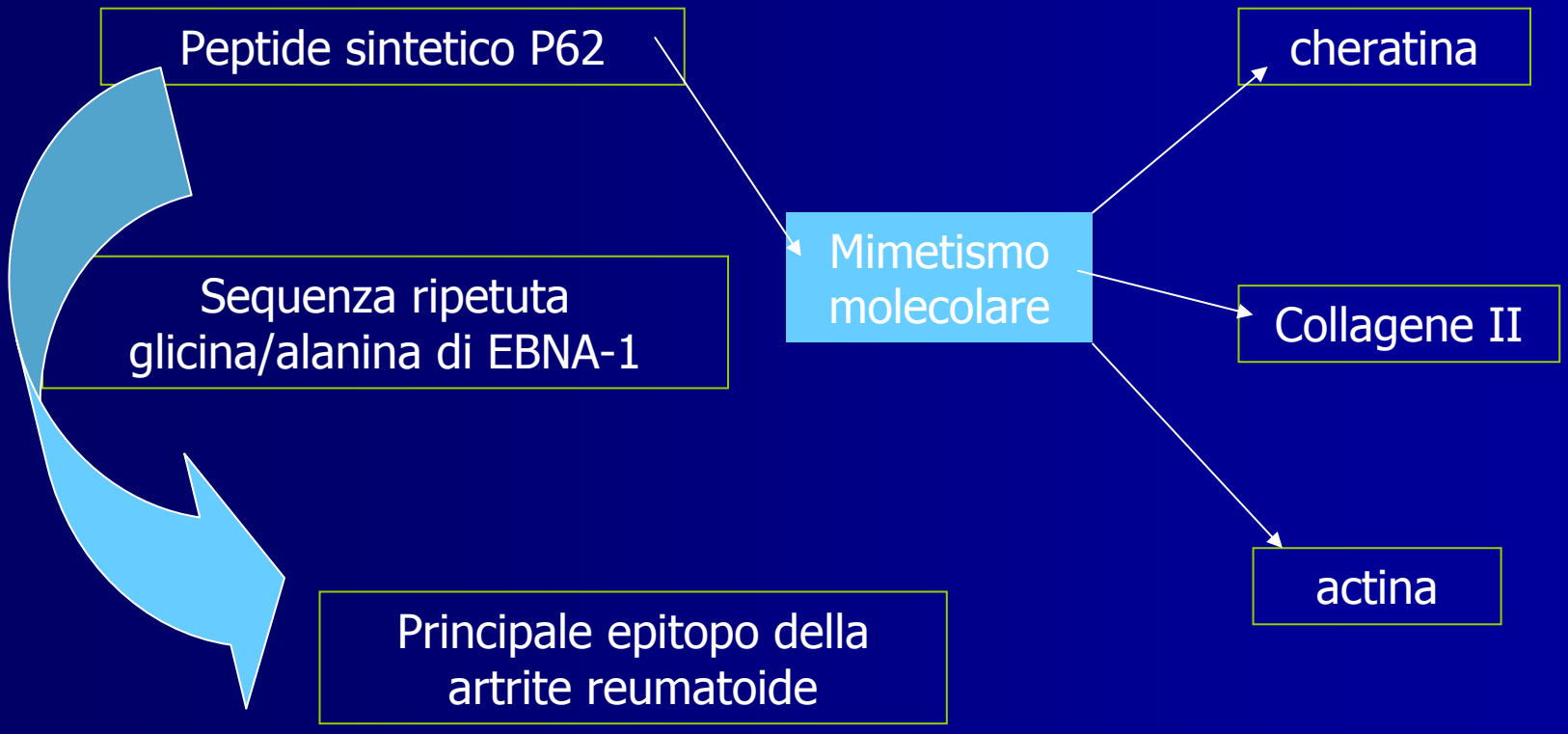
Wildner G, et al. Induction of arthritis and uveitis in Lewis rats by antigenic mimicry of peptides from HLA-B27 and cytokeratin. Eur J Immunol. 2002 Jan;32(1):299-306.



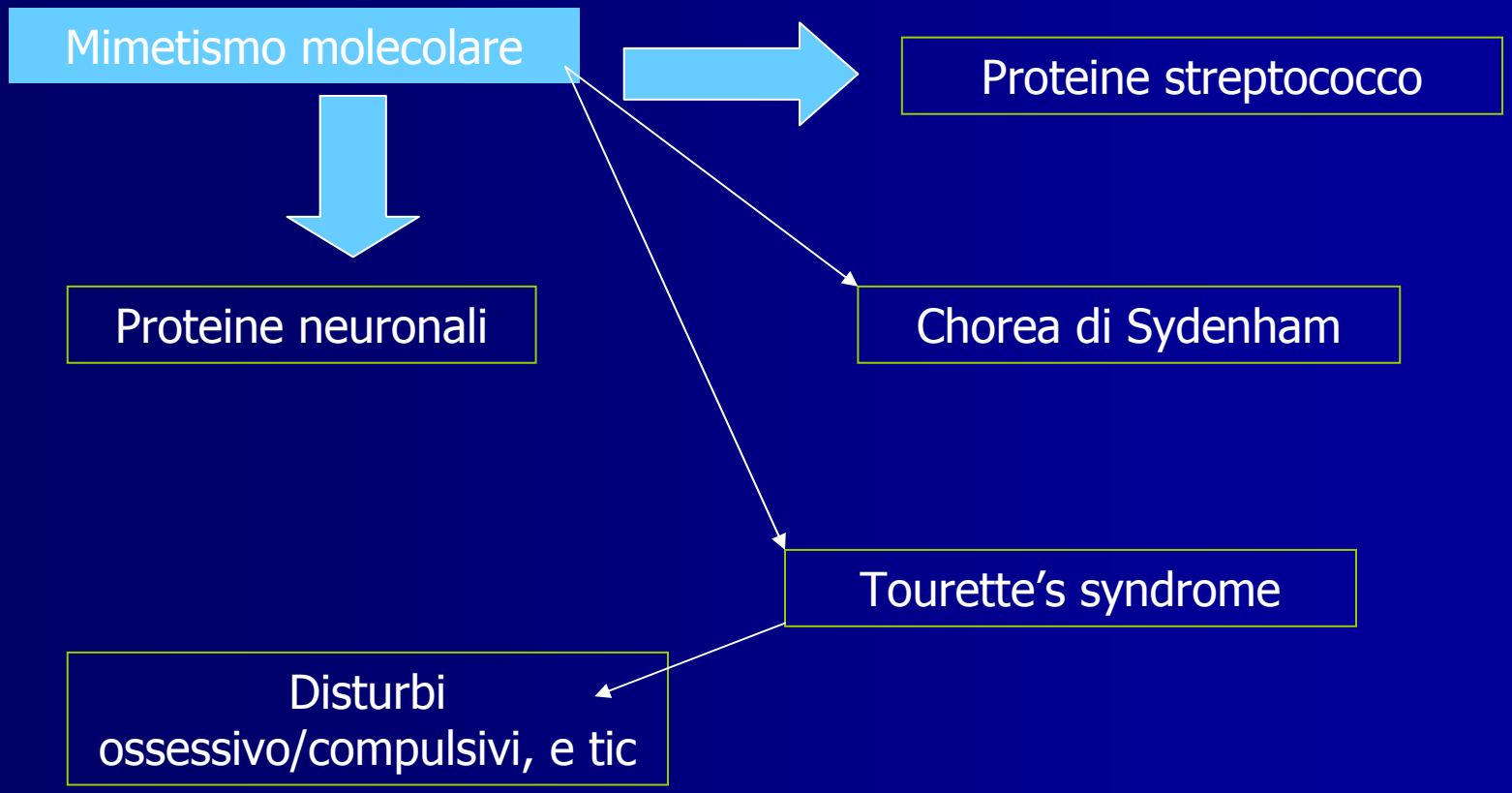
Rambukkana A, et al. Mycobacterial 65,000 MW heat-shock protein shares a carboxy-terminal epitope with human epidermal cytokeratin 1/2. Immunology. 1992 Oct;77(2):267-76.



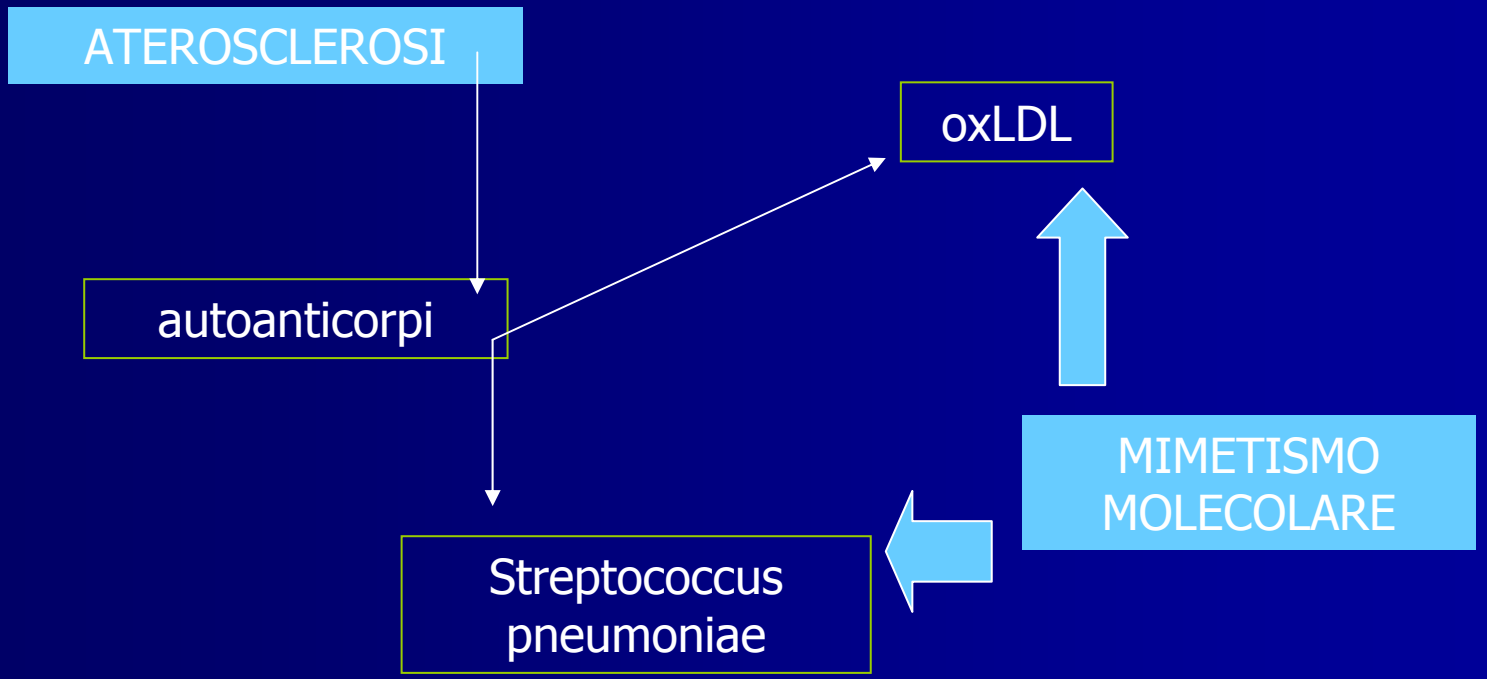
Baboonian C, et al. Cross reaction of antibodies to a glycine/alanine repeat sequence of Epstein-Barr virus nuclear antigen-1 with collagen, cytokeratin, and actin. Ann Rheum Dis. 1991 Nov;50(11):772-5.



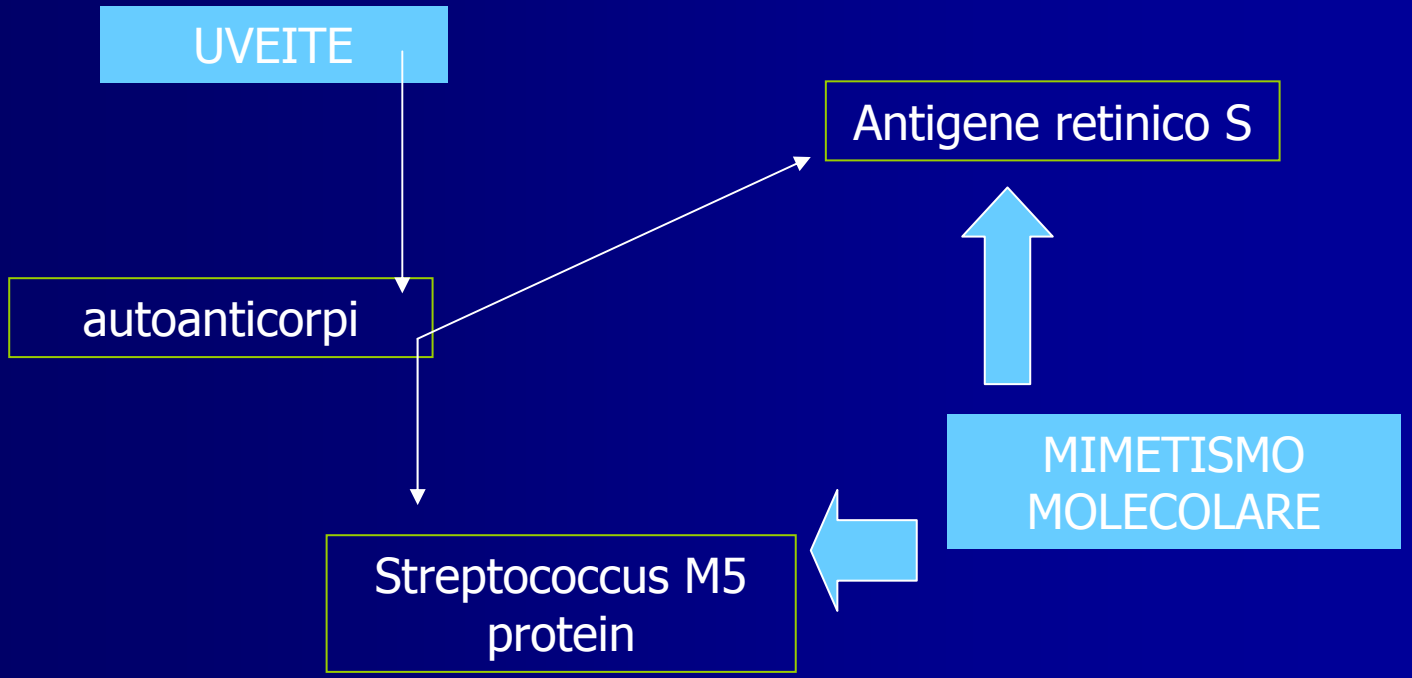
Dale RC. [Streptococcus pyogenes and the brain: living with the enemy]. Rev Neurol. 2003 Jul 1-15;37(1):92-7.



Binder CJ, et al. Pneumococcal vaccination decreases atherosclerotic lesion formation: molecular mimicry between Streptococcus pneumoniae and oxidized LDL. Nat Med. 2003 Jun;9(6):736-43. Epub 2003 May 12.



Lerner MP, et al. Immunological mimicry between retinal S-antigen and group A streptococcal M proteins. Autoimmunity. 1995;22(2):95-106.



Zilliox AP, et al. **J Clin Immunol. 1993 Nov;13(6):415-23.** Henoch-Schoenlein purpura due to streptokinase.

The syndrome of Henoch-Schoenlein purpura developed in a 74-year-old woman after receiving streptokinase as thrombolytic therapy for an acute myocardial infarction. Renal biopsy revealed mesangial hypercellularity with deposits of IgA. Skin biopsy also revealed IgA deposition. Immunological studies showed evidence of sensitization to streptokinase. Elevated IgG, IgA, IgM, and IgE antistreptokinase antibodies were detected in the acute serum. Positive immediate skin reactivity to streptokinase was also present. Serum precipitins to streptokinase disappeared when IgA was removed from the serum. Positive staining with biotinylated streptokinase was seen in the skin in the same pattern of distribution as IgA. These findings strongly support the role of streptokinase and IgA in the pathogenesis of Henoch-Schoenlein purpura in this patient. A control group of streptococcal-infected patients showed no immune response to streptokinase. Another control group of streptokinase-treated patients, who had no untoward reaction, had elevated immunoglobulin classes and precipitins to streptokinase. However, the precipitating antibody was IgG and streptokinase skin tests were negative.